Ukrainian Journal of
Veterinary and Agricultural Sciences
http://ujvas.com.ua

The Stepan Gzhytskyi National University of Veterinary Medicine and Biotechnologies Lviv

Volume 5
Number 1

original article | UDC 636.7.09:616.831 | doi: 10.32718/ujvas5-1.04

Dynamics and diagnosis of severe pneumocephalus of unknown etiology in dog

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Article info
Received 02.02.2022
Received in revised form 03.03.2022
Accepted 04.03.2022

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Abstract

Modern veterinary medicine and its subdivision – veterinary neurology, are constantly evolving. New diagnostic methods are emerging every day, and the list of new, previously unexplored animal diseases is expanding. Much information about the pathogenesis and etiology of diseases is borrowed from human medicine, which is a big problem, because human physiology is different from animals and, as a consequence, the dynamics of the disease are also different. That is why the continuous improvement of veterinary therapy and diagnosis of diseases is relevant and necessary. One of the rare and severe pathologies is pneumocephalus. It is reported that this is a condition in which there is a gradual accumulation of air in the cranial cavity or ventricles of the brain, creating a life-threatening condition. It is considered that pneumocephalus can occur spontaneously but, in most cases, is diagnosed after trauma or surgery with impaired integrity of the skull bones and meninges. This article describes a case of pneumocephalus of unknown etiology in a young dog who suffered a head injury while playing with the owner. During the neurological examination of the animal conducted immediately after the incident was diagnosed with severe central nervous system lesions (sopor, opisthotonus, tetraparesis); magnetic resonance imaging revealed significant brain damage with pronounced ventricular dilatation and the formation of a cavity filled with air. Re-diagnosis after 14 days showed progression of pneumocephalus with gas accumulation in the brain cavities. The contents of the ventricles have a division into liquid (cerebrospinal fluid), and air, progressive inflammatory processes in the brain's parenchyma were not observed; a fistula or canal connecting the cranial cavity and the external environment was not detected. Symptomatic and supportive therapy, carried out during the entire period of the dog's stay in the veterinary clinic, did not have positive results. The animal was euthanized according to medical indications and the owner's wishes.

Keywords: dog, brain, trauma, pneumocephalus, magnetic resonance imaging, myelitis, hematomyelia.

Citation:
Studenok, A. A., & Trokoz, V. A. (2022). Dynamics and diagnosis of severe pneumocephalus of unknown etiology in the dog. *Ukrainian Journal of Veterinary and Agricultural Sciences*, 5(1), 24–30.

1. Introduction

Tensed pneumocephaly is a rare pathology of the central nervous system in animals and humans, which occurs due to the accumulation of gas under pressure in the cranial cavity or brain ventricles. The course of the disease is characterized by a long latent phase and rapid exacerbation of clinical symptoms and, in the absence of proper treatment, leads to death (Andrews & Canalis, 1986; Garosi et al., 2002; Cunqueiro & Scheinfeld, 2018). Pneumocephalus, in most cases, occurs due to disruption of the integrity of the skull bones and meninges after surgery or injury, which leads to the formation of fistulas between nerve tissue and the environment (Cavanaugh et al., 2008; Shea et al., 2018; Kohl et al., 2021; Moral et al., 2021), as well as the possibility of gas-producing infection (*Escherichia coli, Enterobacter cloacae, Klebsiella aerogenes, Aspergillus* spp.) (Tanaka et al., 1989; Fletcher et al., 2006; Launcelott et al., 2016), with the subsequent development of inflammatory processes in the parenchyma of the brain and its meninges (Aksoy et al., 2013). Diagnostic measures were performed on magnetic resonance imaging devices Hitachi AIRIS Vento Open (0.3T) and Philips Intera (1.5T) using scanning modes: T1, T1 with contrast enhancement “Gadovist”, T2, and FLAIR.

The purpose of research is the course of the disease with a description of the main clinical symptoms and characteristic changes in the brain's structure, which MRI can detect.

2. Materials and methods

MRI examination of the brain and spinal cord was performed in commercial veterinary laboratories in Kyiv. The final diagnosis was established based on changes in structures of a parenchyma of the central nervous system characteristic of this pathology. The increase in size and air accumulation in the brain's ventricles is a pathognomonic pattern of pneumocephalus in animals and humans.

All studies were conducted following the Law of Ukraine on protecting animals from cruelty (2012) and the European Convention for the Protection of Pets (2013).

The search for scientific data was performed by analyzing open sources of scientific literature: PubMed and Google Scholar.

3. Results and discussion

3.1. Results

Mixed-breed dog, male, 1,5 years old, body weight 17 kg. He was admitted to the veterinary clinic with acute neurological disorders. According to the owners, the dog, playing, got up on its hind legs and, not keeping his balance, fell on his back and, as a result, received a strong blow to the head on the floor.

The doctor's examination revealed: forced lying down, opisthotonus, disorientation and soporous state of consciousness, neck pain, and tetraparesis. From anamnesis vitae, it is known that a dog at a young age probably had a brain injury due to a car accident. Owners found the dog on the road with soft tissue injuries and neurological disorders. The diagnosis was not performed then; treatment was symptomatic, and the animal fully recovered. Also, six months before the main disease, dog owners went to the veterinary clinic with the animal problem during the last two weeks of uncharacteristic behavior, accompanied by an inability to jump out of bed and concentrate on close objects. Due to the patient's aggressiveness and activity, the doctor could not fully examine him and establish the location of the problem and its nature. The owners admitted that the animal's diet consisted mainly of potatoes and meat, which could lead to a lack of nutrients for the body and, consequently, the emergence of the disease. It was proposed to change the animal's feed to a complete commercial diet for treatment. There were no more appeals to this problem.

Treatment of the patient after the fall and the development of neurological symptoms were as follows: butorphanol 0.9 mg/kg, nalbuphine 5.0 mg, mannitol 0.5 g/kg twice with an interval of 1 hour. Infusion at a constant rate for 12 hours: tiletamine/zolazepam 1.0 mg/kg/h, medetomidine hydrochloride 1.5 μg/kg/h, lidocaine 50.0 μg/kg/h. After 12 hours, the gradual withdrawal of the dog from medical sleep and anesthesia developed new symptoms of the disease, which manifested in excessive vocalization, uncontrolled floating movements, and opisthotonus. It was decided to keep the animal asleep for 18–24 hours using medetomidine hydrochloride 1.5 μg/kg/h and profol 4–6 mg/kg/h. In addition, appropriate therapeutic measures were used to maintain normal body function (parenteral feeding, diuresis monitoring, warming, and prevention of bedsores).

The dog underwent an MRI of the brain and cervical spinal cord one day after the injury. In the images of the spinal cord, the hyperintensive (T2) and isointensive (T1) signals were increased at the level of 2–3 vertebrae. This pattern is characteristic of the acute stage of hemorrhage. The intervertebral disc remained intact and hydrated, and no spinal cord compression was detected. These findings indicate traumatic myelopathy with minor parenchymal bleeding (hematomyelia) (Fig. 1 a, b, c) (Mai, 2018).

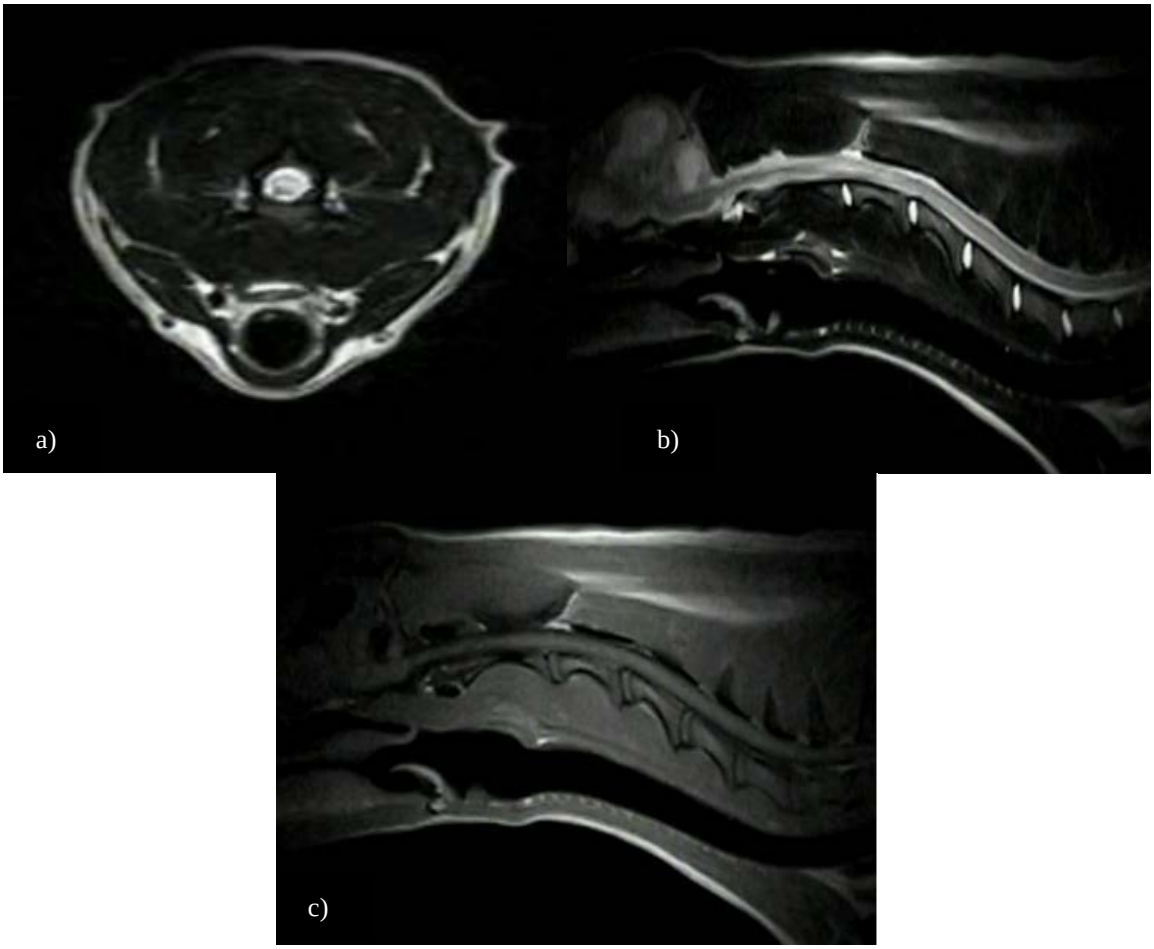

Fig. 1. Magnetic resonance imaging of the dog's spinal cord at the level of 2–3 cervical vertebrae with manifestations of traumatic myelitis/hematomyelia (24 hours after injury)

Note: a – axial section T2; *b* – sagittal section T2; *c* – sagittal section T1 (MRI 0.3T)

Examination of the brain revealed a significant expansion of the left lateral, third and fourth ventricles, protrusion of the frontal lobe of the cerebral cortex in the olfactory bulbs with the formation of granulomas in the lumen of the lattice bone, a cystic cavity filled with gas and located ventrally to the bottom of the third ventricle and dorsally relative to the thalamus (Fig. 2 a, b, c, d). Additionally, edema of the brain parenchyma was registered in the area of the frontal and temporal lobes on the left (Fig. 2 d).

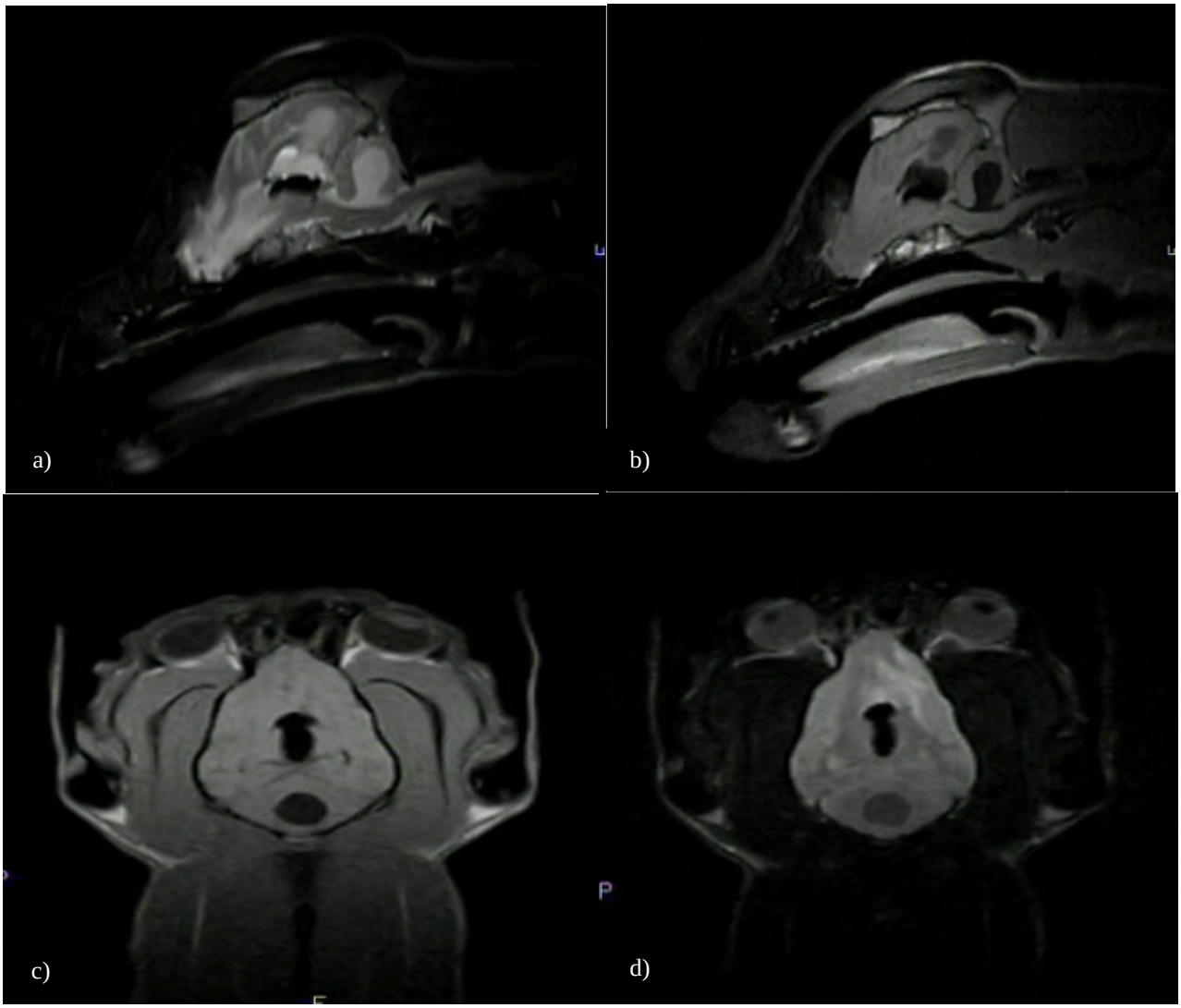

Fig. 2. Magnetic resonance imaging of the dog with post-traumatic changes in the brain (24 hours after injury)
Note: *a, b* – sagittal section at the level of the left lateral ventricle of the brain in T2, T1 modes; *c, d* – coronary section at the level of the fourth ventricle of the brain and cystic cavity in T1, T2 modes (MRI 0.3T)

Note that the accumulation of gas ventrally to the third ventricle cavity was completely separated by a wall from the cerebrospinal fluid and was a full-fledged cystic cavity, formed long before the injury received the day before.

The lesion of the olfactory bulb in its structure is an inflammatory granuloma, probably formed due to a sharp increase in intracranial pressure in the left hemisphere of the brain (Fig. 3 a, b) (Mai, 2018). It is impossible to talk about a frontal bone fracture in the sinus and lattice bone area due to the lack of imaging images of the fracture in the MRI examination. No fistula could connect the ventricular cavity.

The animal's condition deteriorated after 72 hours of symptomatic treatment and medical sleep. The patient developed persistent atony of the gastrointestinal tract (GI tract), infection of the urogenital system, rapidly progressing atrophy of the muscles of the body, and the occurrence of bedsores. Withdrawal of the patient from medical sleep was always accompanied by the restoration of vocalization, uncontrolled floating movements, and self-trauma. Treatment of this condition has focused on restoring gastrointestinal motility, preventing soft tissue and bladder infections, maintaining hydration, and parenteral and partial small-scale enteral nutrition through a gastrostomy.

A repeated MRI was performed on the 14th day after the brain injury. The animal's condition at the time was severe, and the prognosis for survival – was questionable. The examination revealed significant changes in the condition of the ventricles and parenchyma of the organ compared to the previous period. Hyperintensive signal in T2 and FLAIR mode from the cortex of the frontal, temporal areas, and olfactory bulbs decreased significantly; inflammatory granuloma was not visualized (Fig. 4 a, b).

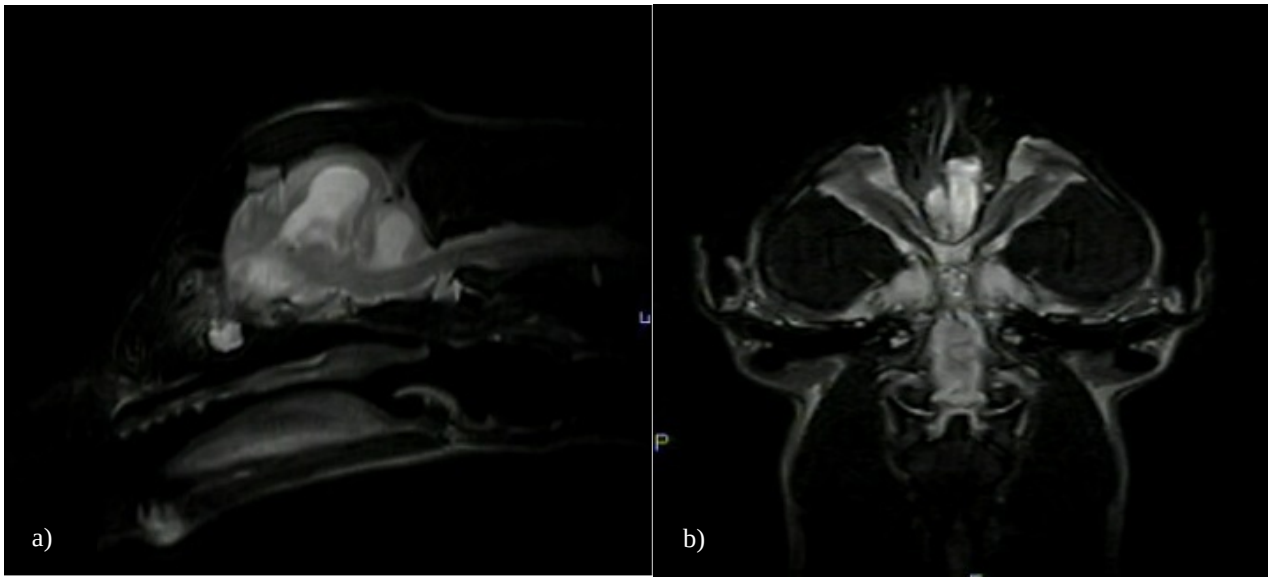

Fig. 3. Magnetic resonance imaging of the dog with post-traumatic changes in the brain (24 hours after injury)
Note: *a, b* – sagittal and coronary sections at the level of olfactory bulb granuloma in T2 mode (MRI 0.3T).

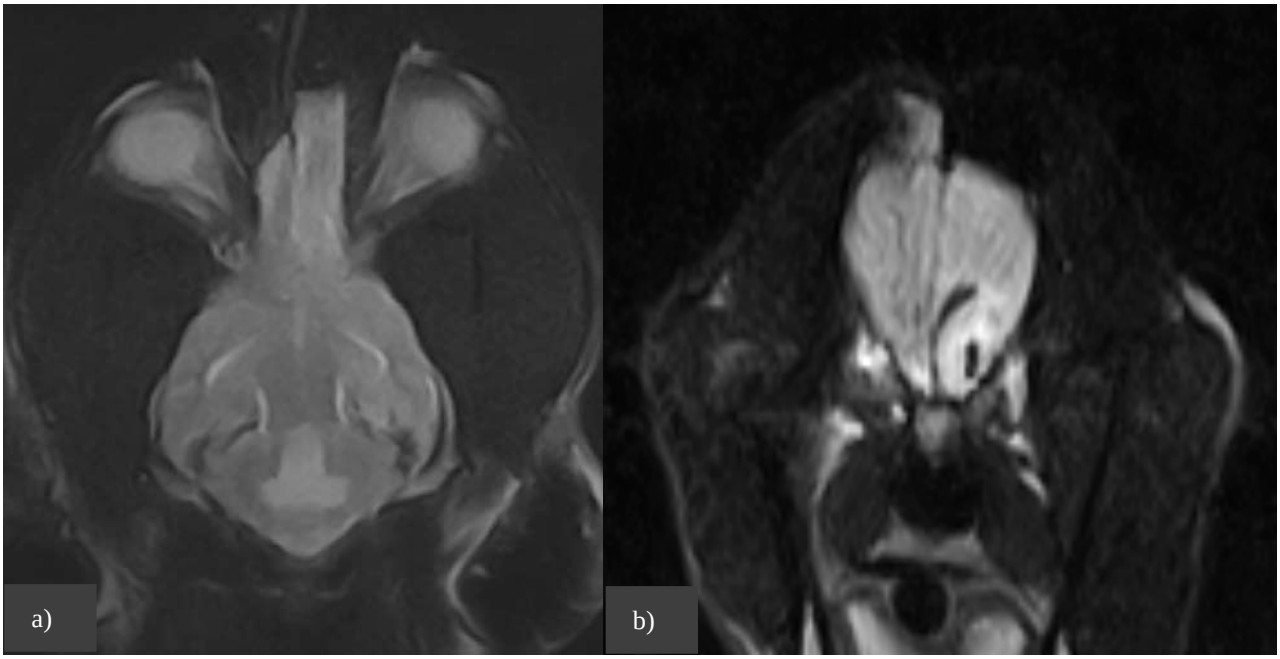

Fig. 4. Magnetic resonance imaging of the dog with post-traumatic changes in the brain (14 days after injury)
Note: *a, b* – coronary, and axial sections in T2 and FLAIR modes show a slight inflammatory process in the left olfactory bulb on the brain parenchyma (MRI 1.5T)

The contents of the ventricles were heterogeneous and were divided into two environments – cerebrospinal fluid and air. The ventricular walls of the brain gave a hyperintensive signal T1, T1 with contrast enhancement, T2, and FLAIR. The meninges and parenchyma of the brain did not accumulate contrast material (Fig. 5 a, b, c).

Reconstruction in 3D format demonstrates the size of the dilated left lateral ventricle and its large-scale nature relative to the brain's parenchyma. It should be noted that pronounced inflammatory processes in the central nervous system were not observed (Fig. 5), which excludes, in our opinion, anaerobic microflora, which could produce gases due to their activities. There is also no duct through which air could enter the ventricles (Fig. 6).

The presented image clearly shows left ventriculomegaly and an ipsilaterally visualized enlarged olfactory bulb, which was affected by increased intracranial pressure.

According to the MRI data and the patient's clinical condition, the pet owners decided to euthanize. No post-mortem autopsy was performed.

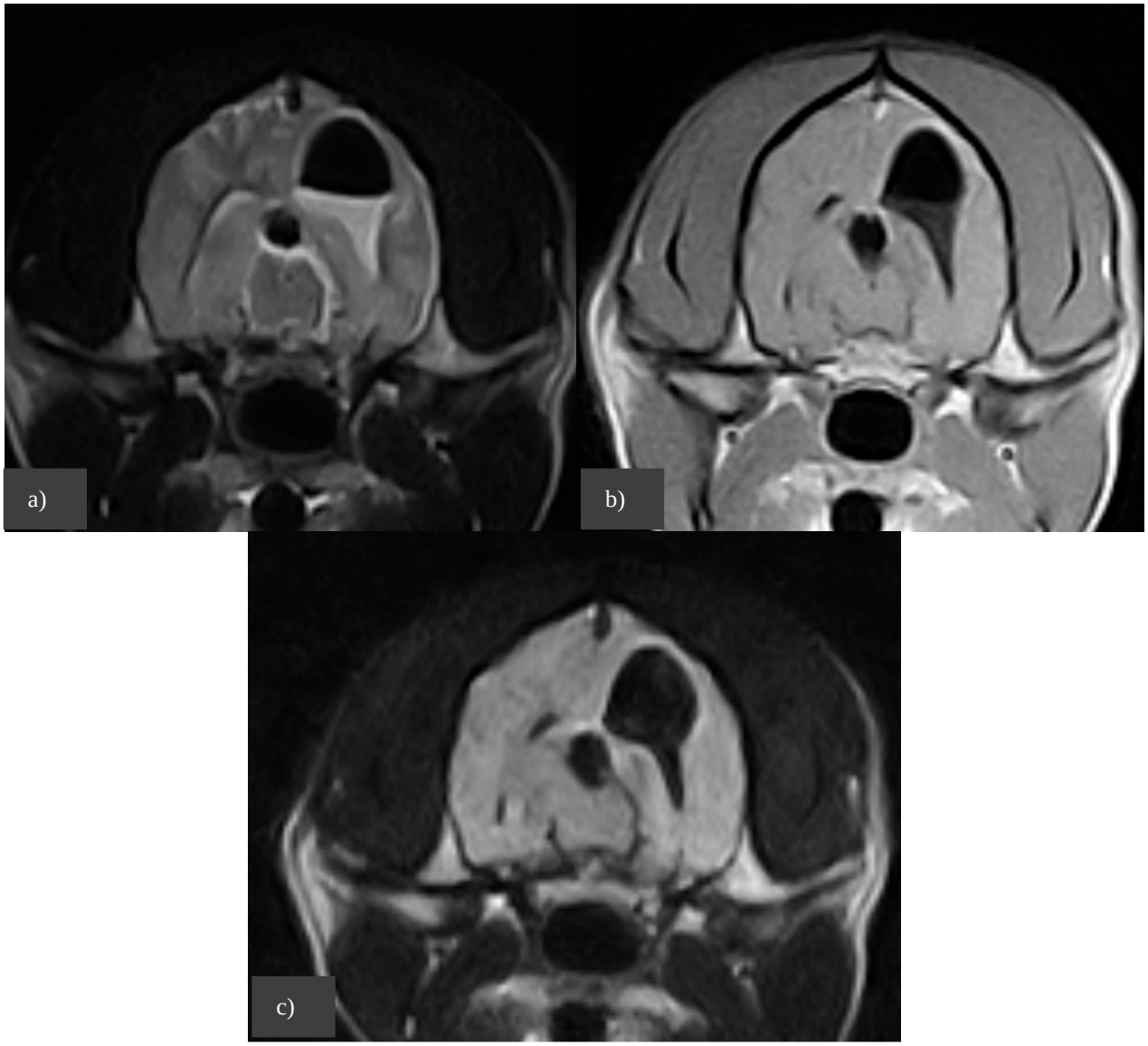

Fig. 5. Magnetic resonance imaging of the dog with post-traumatic changes in the brain (14 days after injury)
Note: *a* – axial section at the level of the lateral and third ventricles in the T2 mode; *b* – axial section at the level of the lateral and third ventricles in the T1 mode with contrast enhancement; *c* – axial section at the level of the lateral and third ventricles in the FLAIR mode

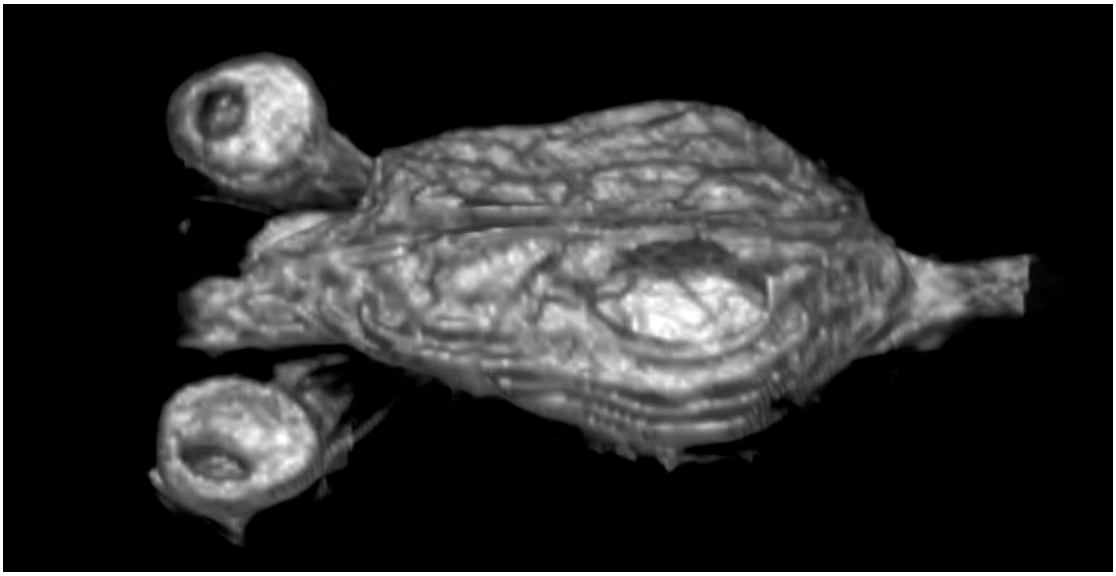

Fig. 6. 3D reconstruction of the dog's brain with post-traumatic changes (14 days after injury)

3.2. Discussions

Comparing the results of the first and subsequent MRI research, we can identify the most characteristic changes that occurred during the development of the disease. In animals, a decrease in intraventricular cerebrospinal fluid was observed with the formation of cavitiesan identical pattern was observed in the first MRI scan, which revealed a cavity in the third ventricle that was formed, most likely during previous trauma, which may be associated with neurological disorders at an early age. A similar pattern is described by the authors Haley & Abramson (2009), who observed a 17-month-old dog with a history of head injury at a young age. Neurological examination and clinical symptoms of the disease were identical and indicated central nervous system damage. According to the results of computed tomography (CT), the authors obtained data on fractures of the left frontal sinus and lattice plate and, as a consequence, the accumulation of air in the lateral, third and fourth ventricles with significant compression of the brain, cerebellum and medulla oblongata (Haley & Abramson, 2009).

Several reports in the scientific and special veterinary literature indicate that the main cause of pneumocephalus in humans and animals (dogs) is a post-traumatic or postoperative complication (Schirmer et al., 2010; Shea et al., 2018; Kohl et al., 2021). According to the collected statistics for 2018, based on international veterinary publications, only 25 % of dogs have pneumocephaly due to injury, and 75 % are associated with postoperative complications (Shea et al., 2018). Spontaneous disease development in human medicine accounts for only 0.6 % of cases (Shea et al., 2018). Other causes of this pathology may also include the growth of gas-producing bacteria (Tanaka et al., 1989; Shea et al., 2018), chronic rhinitis or rhinosinusitis (Sena et al., 2017), barotrauma (Shea et al., 2018), otitis media (Garosi et al., 2002). Gas localization sites include epidural, subdural, subarachnoid, cerebral, and intraventricular spaces (Ros et al., 2015).

To date, two mechanisms of pneumocephalus in violation of the integrity of the skull bones are described: the first theory describes the valvular mechanism in which air is injected into the cranial cavity at the time of coughing, sneezing, or barking, when the air pressure in the sinuses exceeds intracranial; the other is hydrodynamic, has a delayed onset of clinical symptoms and is associated with slow “leakage” or reabsorption of cerebrospinal fluid. As the volume of cerebrospinal fluid gradually decreases, negative pressure is created in the ventricular cavities of the brain, which allows you to replace the lost fluid with air (Sprague & Poulgrain, 1999; Garosi et al., 2005; Haley & Abramson, 2009; Shea et al., 2018).

Treatment of this condition with small amounts of accumulated gas, if the patient does not have nervous system disorders or other disorders, is not required. To prevent further disease development, it is necessary to conduct thorough diagnostic measures to find the causes of pneumocephalus: MRI, CT of the brain and skull bones, rhinoscopy, and diagnosis of infections that could potentially be a source of gas (Garosi et al., 2005). In severe rhinorrhea, it is necessary to differentiate from cerebrospinal fluid leakage by measuring glucose levels, especially in patients with a history of head injury. The test can be performed only in the absence of blood impurities in the secretion or infections in the upper respiratory tract, and the blood glucose content should be below six mmol/l (Shea et al., 2018). In the absence of skull fractures or other causes of gas accumulation and disease progression, the accumulated air can be absorbed on its own within a few weeks. For example, people need up to 6 months to absorb 50 ml of air (Shea et al., 2018).

Surgical treatment of pneumocephalus involves performing a craniotomy and closing the hole with polymethyl acrylate or the patient's tissues – free temporal fascia graft, fat (Garosi et al., 2005). Accumulated gas is removed by puncturing dilated ventricles (Sharma et al., 1989; Garosi et al., 2005; Haley & Abramson, 2009).

4. Conclusions

The occurrence of such a life-threatening condition as severe pneumocephalus is quite rare in veterinary medicine. The main reasons for the development of this pathology of the brain and spinal cord may be a violation of the integrity of the barriers (meninges, skull bones) between the environment and the brain, the development of gas-producing microorganisms, and fungi or, in very rare cases, spontaneously. Treatment for clinical symptoms of the disease is to reduce intracranial pressure and surgical closure of the formed fistula or canal with artificial materials or the patient's own tissues. Practitioners should always keep in mind the symptoms and preconditions of pneumocephalus in animals, which will help to help the patient in time and save his life.

Conflict of interest.

The authors state that there is no conflict of interest.

References

Aksoy, F., Dogan, R., Ozturan, O., et al. (2013). Tension pneumocephalus: an extremely small defect leading to an extremely serious problem. *American Journal of Otolaryngology*, 34(6), 749–752.
[Crossref] [Google Scholar]

Andrews, J. C., & Canalis, R. F. (1986). Otogenic pneumocephalus. *Laryngoscope*, 96(5), 521–528.
[Crossref] [Google Scholar]

Cavanaugh, R. P., Aiken, S. W., & Schatzberg, S. J. (2008). Intraventricular tension pneumocephalus and cervical subarachnoid pneumorrhachis in a bull mastiff dog after craniotomy. *Journal of Small Animal Practice*, 49, 244–248.
[Crossref] [Google Scholar]

Cunqueiro, A., & Scheinfeld, M. H. (2018). Causes of pneumocephalus and when to be concerned about it. *Emergency Radiology*, 25, 331–340.
[Crossref] [Google Scholar]

Fletcher, D. J., Snyder, J. M., Messinger, J. S., Chiu, A. G., & Vite, C. H. (2006). Ventricular pneumocephalus and septic meningoencephalitis secondary to dorsal rhinotomy and nasal polypectomy in a dog. *Journal of the American Veterinary Medical Association*, 229(2), 240–245.
[Crossref] [Google Scholar]

Garosi, L. S., McConnell, F. J., & Lujan, A. (2005). What is your diagnosis? Pneumocephalus. *Journal of the American Veterinary Medical Association*, 226(7), 1057–1058.
[Crossref]

Garosi, L. S., Penderis, J., Brearley, M. J., Brearley, J. C., Dennis, R., & Kirkpatrick, P. J. (2002). Intraventricular tension pneumocephalus as a complication of transfrontal craniectomy: a case report. *Veterinary Surgery*, 31(3), 226–231.
[Crossref] [Google Scholar]

Haley, A. C., & Abramson, C. (2009). Traumatic pneumocephalusin a dog. *Journal of the American Veterinary Medical Association*, 234(10), 1295–1298.
[Crossref] [Google Scholar]

Kohl, S., Köhler, C., & Kiefer, I. (2021). MRI diagnosis of spontaneous intraventricular tension-pneumocephalus in a 10-month-old male Saarloos Wolfdog. *Veterinary radiology & ultrasound*, 63(3), E20–E23.
[Crossref] [Google Scholar]

Kohl, S., Köhler, C., & Kiefer, I. (2021). MRI diagnosis of spontaneous intraventricular tension-pneumocephalus in a 10-month-old male Saarloos Wolfdog. *Veterinary Radiology & Ultrasound*, 63, E20–E23.
[Crossref] [Google Scholar]

Launcelott, Z. A., Palmisano, M. P., Stefanacci, J. D., & Whitney, B. L. (2016). Ventricular pneumocephalus, cervical subarachnoid pneumorrhachis, and meningoencephalitis in a dog follow in rhinotomy for chronic fungal rhinitis. *Journal of the American Veterinary Medical Association*, 248(4), 430–435.
[Crossref] [Google Scholar]

Moral, M., Blanco, C., Martínez, J., & Lorenzo, V. (2021). Delayed traumatic pneumocephalus and cervical pneumorrhachis in a dog. *Veterinary Record Case Reports*, 9, 3.
[Crossref] [Google Scholar]

Ros, C., dela Fuente, C., Novellas, R., & Añor, S. (2015). What is your diagnosis? Pneumocephalus. *Journal of the American Veterinary Medical Association*, 246(9), 959–961.
[Crossref]

Schirmer, C. M., Heilman, C. B., & Bhardwaj, A. (2010). Pneumocephalus: case illustrations and review. *Neurocrit Care*, 13, 152–158.
[Crossref] [Google Scholar]

Sena, J. O., Costa, K. C. R., Costa, P. M., Miranda, F. G., Silva, J. F., & Tôrres, R. C. S. (2017). Intraventricular pneumocephalus associated with nasocephalic necrosisin a puppy: a case report. *Arquivo Brasileirode Medicina Veterinária e Zootecnia*, 69(2), 333–339.
[Crossref] [Google Scholar]

Sharma, B. S., Tewari, M. K., Khosla, V. K., Pathak, A., & Kak, V. K. (1989). Tension pneumocephalus following evacuation of chronic subdural haematoma. *British journal of neurosurgery*, 3(3), 381–387.
[Crossref] [Google Scholar]

Shea, A., Dominguez, E., & Stewart, J. (2018). Spontaneous non-traumatic tension pneumocephalus in a dog with a nasal meningoencephalocele. *Veterinary Record Case Reports*, 6(4), e000670.
[Crossref] [Google Scholar]

Sprague, A., & Poulgrain, P. (1999). Tension pneumocephalus: a case report and literature review. *Journal of clinical neuroscience*, 6(5), 418–420.
[Crossref] [Google Scholar]

Tanaka, T., Takagi, D., Takeyama, N., & Kitazawa, Y. (1989). “Spontaneous” pneumocephalus associated with aerobic bacteremia. *Clinical imaging*, 13(2), 134–139.
[Crossref] [Google Scholar]

Mai, W. (2018). *Diagnostic MRI in dogs and cats*. Boca Raton: CRC Press.
[Google Scholar]